omas, and is limited to the cranial–spinal axis, without systemic disease. A known high-risk group for PCNSL comprises patients with immunosuppression, but in the last 15–20 years there has been a threefold increase in patients who are immunocompetent, and the causative factors for this increase are unknown [1–3]. PCNSL commonly presents as a rapidly space-occupying lesion. Rarely, patients without obvious brain parenchymal tumors present with only primary meningeal involvement [1–3]. Our case has remarkable similarities with the nine cases of PLML reported by Lachance et al [4]. These authors believe that PLML is a distinct clinical entity. Abnormal CSF is present in 80% of PCNSL cases with hypoglycorrhachia, increased protein levels or pleocytosis. It has been stated that finding malignant cells in the CSF is rare, with an incidence of 10–25% [1–4].

In a patient with pleocytosis, the first cause suggested will usually be central nervous system infection. Our patient also had remarkable similarities with chronic meningitis in his symptoms and the findings of physical and CSF examinations. However, other causes of pleocytosis must be remembered too, and PCNSL, or PLML, has therefore to be added to the differential diagnosis of chronic meningitis.

Yasemin Heper[1], *Resit Mistik*[1], *Omer Yerci*[2], *Figen Altintas*[1], *Halis Akalin*[1]
[1]Department of Clinical Microbiology and Infectious Diseases, Uludag University, School of Medicine, Görükle, Bursa, Turkey;
[2]Department of Pathology, School of Medicine, Uludag University, Görükle, Bursa, Turkey

Accepted 21 July 1997

References

1. Fine HA, Mayer JM. Primary central nervous system lymphoma. Ann Intern Med 1993; 119: 1093–104.
2. Miller DC, Hochberg FH, Harris NL, Gruber ML, Louis DN, Cohen H. Pathology with clinical correlations of primary central nervous system non-Hodgkin's lymphoma. Cancer 1994; 74: 1383–97.
3. Parekh HJ, Sharma RR, Lynch PG, Keogh AJ, Probhu SS. Primary cerebral lymphoma. Report of 24 patients and review of the literature. Br J Neurosurg 1992; 6: 563–73.
4. Lachance DH, O'Neill BP, Macdonald DR, et al. Primary leptomeningeal lymphoma: report of 9 cases, diagnosis with immunocytochemical analysis, and review of the literature. Neurology 1991; 41: 95–100.

Corynebacterium pseudotuberculosis infection in a butcher

Clin Microbiol Infect 1997; 3: 696–698

Infections due to *Corynebacterium pseudotuberculosis* are frequent in sheep and goats. However, infections due to this microorganism are rare in humans. To our knowledge, there have been only 13 cases described in the literature [1–11]. Since cultures growing *Corynebacterium* species are frequently considered to be contaminated, it is conceivable that the incidence of this disease is underestimated in human beings.

A 30-year-old Turkish man attended the surgical outpatient clinic because of a painful epitrochlear swelling on his left arm which had been present for 1 month. His past medical history was unremarkable. He denied ever having had venereal disease or venereal exposure. He had immigrated from Turkey to Switzerland 2 months earlier and he was unemployed. In Turkey, he had worked as a sheep rancher and butcher. He could not remember any previous trauma. His parents and his 11 sisters and brothers were living and healthy. The patient was afebrile and did not report symptoms of systemic disease. Clinical examination showed a firm, large (7×5 cm), moderately tender, non-inflamed, non-fluctuant swelling on his left arm which was attached to the humerus. In addition, there was a large (3 cm) tender axillary lymph node. The laboratory work-up revealed normal hematologic values except for a leukocytosis of 12×10^9/L. The C-reactive protein was 11 mg/L. An HIV screening test was negative. A soft tissue swelling but normal bone structure were seen in radiographs of the upper arm. Magnetic resonance imaging showed a mass of 7×4×3 cm between the triceps and biceps muscles infiltrating the muscle and the neurovascular bundle. This finding was interpreted as either abscess or sarcoma. With this differential diagnosis, a biopsy was performed but the operation revealed an abscess, which was drained. A total excision was not feasible because of infiltration of the muscle, vessels and nerve. Gram stain of the pus showed polymorphonuclear leukocytes, but no microorganisms. Two tissue samples grew *C. pseudotuberculosis* within 5 days. This isolate was initially interpreted as a contaminant. When the histology (see below) became available, it was considered to be the causative agent. Oral antimicrobial therapy with clarithromycin (500 mg b.i.d) was started for 2 weeks. After 3 weeks, the wound had healed and the axillar lymph node had become smaller. Three months later, the patient was readmitted to the hospital because of a recurrent large painful epitrochlear swelling on his left arm with erythema around the scar and a large fluctuant axillary node. He denied other symptoms.

Table 1 Human cases with *Corynebacterium pseudotuberculosis* infection

Reference	Sex, age	Exposition	Clinical presentation	Treatment	Complication	Duration
1	M,37	Grass cutter	Malaise, inguinal lymphadenopathy myalgia, hepatomegaly	Tetracycline, excision	None	5 weeks
2	M,28	Sheep rancher	Inguinal lymphadenopathy, painful calf lesion	Penicillin, probenecid, excision, drainage	Sinus tract, relapse of abscess	1 year
3	M,24	Sheep shearer	Asymptomatic axillary lymphadenopathy, sinus tract after puncture	Penicillin, excision	Sinus tract	9 months
4	M,23	Butcher	Tender axillary lymphadenopathy, fever, hepatosplenomegaly	Terramycin, excision	Sinus tract	8 weeks
5	M,20	Sheep rancher	Axillary lymphadenopathy	Excision	None	'Many months'
5	M,40	Rural worker	Inguinal lymphadenopathy	Tetracycline, incision	Sinus tract, secondary wound infection	Not reported
5	F,50	Housewife with contact with sheep	Asymptomatic cervical lymphadenopathy	Antibiotics, excision	Not reported	'Long period'
6	M,21	Silo worker, previously abattoir worker	Painful axillary lymphadenopathy	Cloxacillin, excision	Sinus tract	3 months
7	M,28	Veterinary student	Malaise, dry cough, fever, eosinophilic pneumonia	Erythromycin	None	2 months
8	M,?	Shepherd	Axillary lymphadenopathy	Excision	Sinus tract	3 months
9	M,30	Raw milk ingestion	Cervical lymphadenopathy myalgias, arthralgias	Excision, drainage, erythromycin	Sinus tract, relapse of abscess	3 months
10	M,18	Butcher	Tender epitrochlear lymphadenopathy, erythema, fever	Excision, penicillin, flucloxacillin, tetracycline	Sinus tract, relapse of abscess	6 months
11	M,29	Sheep rancher	Asymptomatic lymphadenopathy	Antibiotic	Sinus tract, secondary wound infection	8 months
Present case	M,30	Butcher, sheep rancher	Tender epitrochlear and axillary lymphadenopathy	Excision, clarithromycin	Sinus tract, relapse of abscess	8 months

He had a leukocytosis of 11×10^9/L and a C-reactive protein of 10 mg/L. The day after admission, spontaneous discharge of the epitrochlear swelling occurred. Surgical drainage was performed. Because of the adjacent neurovascular bundle, full excision was precluded. The fluctuant axillary node was not excised. The culture of the pus and tissue samples again grew *C. pseudotuberculosis*, still susceptible to clarithromycin. Another 6-week course of oral clarithromycin was given. The wound healed within 4 weeks. Follow-up after 6 months revealed no signs of infection, and the patient was in good condition.

All five specimens from both surgical interventions grew pure cultures of anaerobic, Gram-positive coryneform rods which were non-motile, catalase positive, fermentative and non-lipophilic. A conventional biochemical identification scheme and the API Coryne system (bioMérieux, la Balme-les-Grottes, France) showed acid production from glucose and maltose and hydrolysis of urea. Nitrate reduction, esculin hydrolysis and fermentation of sucrose, mannitol and xylose were negative. The identification of *C. pseudotuberculosis* was further supported by a reverse CAMP reaction and the analysis of the cellular fatty acid pattern by means of the Sherlock system (MIDI Inc., Newark, Del, USA). Disk diffusion sensitivity testing showed the organism to be susceptible to penicillin, amoxicillin, cefazolin, cefamandole, ceftriaxone, erythromycin and ciprofloxacin, but resistant to oxacillin and the aminoglycosides. MICs determined by E-test for clarithromycin, ampicillin and ciprofloxacin were <0.016 mg/L, 0.125 mg/L and 0.012 mg/L, respectively. These values did not differ between the isolates obtained at the first and the second operation. The biopsies showed granulomatous, necrotizing non-specific inflammation without signs of malignancy. In addition, there was

non-specific vasculitis and a lymphoplasmacellular inflammation with histiocytes, Langhans' giant cells and epithelioid cell granulomas. Some areas showed marked infiltrates of eosinophilic granulocytes and focal necroses. No microorganisms could be detected.

In sheep and goats, *C. pseudotuberculosis* (formerly called *C. ovis*) commonly causes caseous lymphadenitis [7,12]. The disease is economically important, causing about a 5% shortfall in Australian wool production [12]. Lopez described the first human case in 1966 [1]. To our knowledge, only 13 cases have been published [1–11]. Eight of them were observed in Australia [2–6,10], two in the USA [7,9], and one each in Panama [1], France [8] and New Zealand [11]. Eight of them report close contact with sheep or work as a butcher [2–7,9,11,12]. The present case can be considered as imported from Turkey, and had a classical risk exposure of close contact with sheep.

All published cases had suppurative lymphadenitis (Table 1), except the one described by Keslin et al [7]. This patient had an eosinophilic pneumonia. He was a veterinary student who worked with *C. pseudotuberculosis*. He recovered with a 2 week course of erythromycin. Interestingly, our case, as well as some of the published ones, showed eosinophilic infiltrates in the histology of the lymph nodes.

In most of the published cases with suppurative lymphadenitis, prolonged courses with sinus formation and relapsing abscesses were the rule. Therefore, recurrent surgical interventions for drainage or excision were often required. In none of the cases was antibiotic therapy alone successful. This may be because *C. pseudotuberculosis* is a facultative intracellular pathogen multiplying in macrophages. It escapes the host defense and continues to multiply in the phagolysosomes [13,14]. Cell death and release of bacteria leads to necrotic lesions and to the formation of a thick collagen capsule [15]. Combination of surgery with antimicrobial therapy was successful in all published cases. A prolonged course of intracellularly active antibiotics is needed. In our case, a 2-week course was not long enough. Although *C. pseudotuberculosis* is susceptible to most antibiotics in vitro, the high intracellular concentration of newer macrolides may be advantageous.

This case demonstrates that in granulomatous lymphadenitis, *Corynebacterium* species should be considered as possible pathogens rather than contaminants. An eosinophilic infiltrate should suggest the possibility of infection due to *C. pseudotuberculosis*.

Thomas Bregenzer[1], *Reno Frei*[2]
Helmut Ohnacker[3], *Werner Zimmerli*[1]
[1]Division of Infectious Diseases,
University Hospitals Basel,
Switzerland;
[2]Bacteriology Laboratory,
University Hospitals Basel,
Switzerland;
[3]Institute of Pathology,
University Hospitals Basel,
Switzerland

References

1. Lopez JF, Wong FM, Quesada J. *Corynebacterium pseudotuberculosis*. First case of human infection. Am J Clin Pathol 1966; 46: 562–7.
2. Rountree PM, Carne HR. Human infection with an unusual corynebacterium. J Pathol Bacteriol 1967; 94: 19–27.
3. Hamilton NT, Perceval A, Aarons BJ, Goodyear JE. Pseudotuberculous axillary lymphadenitis caused by *Corynebacterium pseudotuberculosis*. Med J Aust 1968; 2: 356–61.
4. Battey YM, Tonge JI, Horsfall WR, McDonald IR. Human infection with *Corynebacterium ovis*. Med J Aust 1968; 2: 540–3.
5. Blackwell JB, Smith FH, Joyce PR. Granulomatous lymphadenitis caused by *Corynebacterium ovis*. Pathology 1974; 6: 243–9.
6. Henderson A. Pseudotuberculous adenitis caused by *Corynebacterium pseudotuberculosis*. J Med Microbiol 1979; 12: 147–9.
7. Keslin MH, McCoy EL, McCusker JJ, Lutch JS. *Corynebacterium pseudotuberculosis*. A new cause of infectious and eosinophilic pneumonia. Am J Med 1979; 67: 228–31.
8. Peloux Y, Marexca C, Oddou JH. Suppurative lymphadenitis caused by *Corynebacterium pseudotuberculosis*—report of a case observed in an alpine shepherd. Nouv Presse Med (Paris) 1980; 42: 3182–4.
9. Goldberger AC, Lipsky BA, Plorde JJ. Suppurative granulomatous lymphadenitis caused by *Corynebacterium ovis* (*pseudotuberculosis*). Am J Clin Pathol 1981; 76: 486–90.
10. Richards M, Hurse A. *Corynebacterium pseudotuberculosis* abscesses in a young butcher. Aust NZ J Med 1985; 15: 85–6.
11. House RW, Schousboe M, Allen JP. *Corynebacterium ovis* (*pseudotuberculosis*) lymphadenitis in a sheep farmer: a new occupational disease in New Zealand. NZ Med J 1986; 99: 659–62.
12. Paton MW, Rose IR, Hart RA, et al. New infection with *Corynebacterium pseudotuberculosis* reduces wool production. Aust Vet J 1994; 71: 47–9.
13. Hard GC. Examination by electron microscopy of the interaction between peritoneal phagocytes and *Corynebacterium ovis*. J Med Microbiol 1972; 5: 483–91.
14. Hard GC. Comparative toxic effect of the surface lipid of *Corynebacterium ovis* on peritoneal macrophages. Infect Immun 1975; 12: 1439–49.
15. Walker J, Jackson HJ, Eggleton DG, Meeusen ENT, Wilson MJ, Brandon MR. Identification of a novel antigen from *Corynebacterium pseudotuberculosis* that protects sheep against caseous lymphadenitis. Infect Immun 1994; 62: 2562–7.